=> fil ca; d que 17; d que 19; fil medl; d que 135 FILE 'CA' ENTERED AT 16:22:01 ON 14 SEP 94 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 1994 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1967 - 3 Sep 1994 (940903/ED) VOL 121 ISS 10

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L1	29 SEA FILE=CA BENDIG M?/AU
L2	2 SEA FILE=CA LEGER O?/AU
L3	40 SEA FILE=CA SALDANHA J?/AU
L4	1399 SEA FILE=CA JONES S?/AU
L6	311 SEA FILE=CA VLA(W)4
L7	O SEA FILE=CA (L1 OR L2 OR L3 OR L4) AND L6
L1	29 SEA FILE=CA BENDIG M?/AU
L2	2 SEA FILE=CA LEGER O?/AU
L3	40 SEA FILE=CA SALDANHA J?/AU
L4	1399 SEA FILE=CA JONES S?/AU
L8	1009 SEA FILE=CA LEUKOCYTE ADHESION
L9	O SEA FILE=CA (L1 OR L2 OR L3 OR L4) AND L8
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FILE 'MEDLINE' ENTERED AT 16:22:03 ON 14 SEP 94

FILE LAST UPDATED: 9 SEP 1994 (940909/UP). FILE COVERS 1966 TO DATE. +QLF/CT SHOWS YOU THE ALLOWABLE QUALIFIERS OF A TERM.

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** If you post-qualify L-numbered answer sets with limiters **

** (e.g., L4/MAJ), you MUST have highlighting set ON when **

** creating the initial L-numbered answer set to ensure full **

** recall. Type D SET HIGH at an arrow prompt to verify **

** your current setting for highlighting. **
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L29
28 SEA FILE=MEDLINE BENDIG M?/AU
L30
6 SEA FILE=MEDLINE LEGER O?/AU
L31
43 SEA FILE=MEDLINE SALDANHA J?/AU
L32
1636 SEA FILE=MEDLINE JONES S?/AU
L33
321 SEA FILE=MEDLINE VLA(W) 4
L34
436 SEA FILE=MEDLINE RECEPTORS, VERY LATE ANTIGEN+NT/CT
L35
1 SEA FILE=MEDLINE (L29 OR L30 OR L31 OR L32) AND (L33 OR L
34)
```

=> d all 135

L35 ANSWER 1 OF 1 MEDLINE 1994 AN 94266969 MEDLINE Molecular Sequence Data Protein Binding

Receptors, Very Late Antigen: IM, immunology \*Receptors, Very Late Antigen: ME, metabolism

Recombinant Proteins: ME, metabolism

Sequence Homology, Amino Acid Structure-Activity Relationship

0 (integrin alpha4beta1); 0 (vascular cell adhesion molecule); 0
(Antibodies, Monoclonal); 0 (Antigenic Determinants); 0 (Cell
Adhesion Molecules); 0 (Chimeric Proteins); 0 (IgG); 0
(Immunoglobulins, Surface); 0 (Integrins); 0 (Receptors, Very Late
Antigen); 0 (Recombinant Proteins)

CN

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2907 SEA FILE=MEDLINE HUMANI?
L40
              O SEA FILE=MEDLINE (L33 OR L34) AND L40
L41
L39
           249 SEA FILE=MEDLINE 21(W)6
          85488 SEA FILE=MEDLINE IMMUNOGLOBULIN#
L44
L45
              O SEA FILE=MEDLINE L39(5A)L44
L39
           249 SEA FILE=MEDLINE 21(W)6
          2907 SEA FILE=MEDLINE HUMANI?
L40
L46
        O SEA FILE=MEDLINE L39 AND L40
L33
           321 SEA FILE=MEDLINE VLA(W)4
           436 SEA FILE=MEDLINE RECEPTORS, VERY LATE ANTIGEN+NT/CT
L34
L47
          12912 SEA FILE=MEDLINE MULTIPLE SCLEROSIS+NT/CT
L48
              1 SEA FILE=MEDLINE (L33 OR L34) AND L47
          2907 SEA FILE=MEDLINE HUMANI?
L40
           5596 SEA FILE=MEDLINE CELL ADHESION MOLECULES+NT/CT
L49
              4 SEA FILE=MEDLINE L40 AND L49
L50
           436 SEA FILE=MEDLINE RECEPTORS, VERY LATE ANTIGEN+NT/CT
L34
L51
            895 SEA FILE=MEDLINE CHIMERIC PROTEINS+NT/CT
              7 SEA FILE=MEDLINE L51 AND L34
L52
         389778 SEA FILE=MEDLINE MICE+NT/CT
L53
              3 SEA FILE=MEDLINE L52 AND L53
L54
            7 (L48 OR L50 OR L54) NOT (L35) previously
L56
FILE 'CA' ENTERED AT 16:26:10 ON 14 SEP 94
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=> dup rem 156,155FILE 'MEDLINE' ENTERED AT 16:26:10 ON 14 SEP 94

USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 1994 AMERICAN CHEMICAL SOCIETY (ACS) PROCESSING COMPLETED FOR L56 PROCESSING COMPLETED FOR L55 11 DUP REM L56 L55 (1 DUPLICATE REMOVED) L57

=> d bib ab 157 1-11; fil hom

ANSWER 1 OF 11 MEDLINE 1994 L57

AN 94193647 MEDLINE

Identification of putative ligand binding sites within I domain of ΤI integrin alpha 2 beta 1 (VLA-2, CD49b/CD29).

Kamata T; Puzon W; Takada Y ΑU

CS Department of Vascular Biology, Scripps Research Institute, La Jolla, California 92037.

NC GM47157 (NIGMS)

SO J Biol Chem, (1994 Apr 1) 269 (13) 9659-63. Journal code: HIV. ISSN: 0021-9258.

CY United States

acids found in both domain 1 and 4 were required for VLA-4 binding to either domain. Five of these amino acids represent a conserved motif also found in ICAM domains. We propose that integrin binding to these Iq-like domains depends on residues within this conserved motif. Specificity of integrin binding to Ig-like domains may be regulated by a set of nonconserved residues distinct from the conserved motif.

- L57 ANSWER 3 OF 11 MEDLINE 1994
- AN 94168259 MEDLINE
- Monoclonal antibodies -- immunotherapy for the critically ill. ΤI
- AU
- Renal Department, Queen Elizabeth Hospital, Woodville, South CS Australia.
- Anaesth Intensive Care, (1993 Dec) 21 (6) 739-51. Ref: 175 SO Journal code: 4M5. ISSN: 0310-057X.
- CY Australia
- Journal; Article; (JOURNAL ARTICLE) DT General Review; (REVIEW) (REVIEW, TUTORIAL)
- LA English
- Priority Journals; Nursing Journals FS
- EM
- Monoclonal antibodies (mAb) have revolutionised many areas of AB medicine, particularly research and diagnostics. Murine, human and humanized mAb have all been developed. The most important clinical applications to date have been in the fields of transplantation and oncology. Experimental and limited clinical trials suggest mAb are emerging as a new therapeutic strategy in the critically ill. Antibodies against a variety of bacteria or their products are potentially useful in gram-positive and gram-negative shock. Anti-cytokine and anti-neutrophil adhesion molecule mAb may be effective not only in septic shock but also in other conditions associated with acute inflammation and cytokine release, e.g., acid aspiration, ischaemia/reperfusion injury (myocardial infarction, haemorrhagic shock, aortic aneurysm repair). Antibodies inhibiting neutrophil adhesion may also be efficacious in asthma, pulmonary fibrosis, meningitis and cerebral malaria. The use of these and other mAb in intensive care is an exciting prospect and future clinical studies will determine the extent of their role in the management of the critically ill.
- ANSWER 4 OF 11 MEDLINE 1994 L57
- AN 93305386 MEDLINE
- Regulation of HIV production by blood mononuclear cells from TI HIV-infected donors: II. HIV-1 production depends on T cell-monocyte interaction.
- Diegel ML; Moran PA; Gilliland LK; Damle NK; Hayden MS; Zarling JM; AU Ledbetter JA
- Bristol-Myers Squibb Pharmaceutical Research Institute, Seattle, WA CS 98121.
- NC RO1 AI 28065 (NIAID)
- AIDS Res Hum Retroviruses, (1993 May) 9 (5) 465-73. SO Journal code: ART. ISSN: 0889-2229.
- United States CY
- Journal; Article; (JOURNAL ARTICLE) DT
- English LA

MBP to brain ECs but that adhesion mols. other than VLA-4/VCAM-1 are involved because anti-VLA-4 and anti-VCAM-1 did not produce complete inhibition.

ANSWER 6 OF 11 MEDLINE 1994 L57

DUPLICATE 1

Page 9

AN 93308213 MEDLINE

TI Dual expression of CD45RA and CD45RO isoforms on myelin basic protein-specific CD4+ T-cell lines in multiple sclerosis.

Qin Y; Van Den Noort S; Kurt J; Gupta S AU

Department of Medicine, University of California, Irvine 92717. CS

NC AI-26456 (NIAID)

SO J Clin Immunol, (1993 Mar) 13 (2) 152-61. Journal code: HRC. ISSN: 0271-9142.

CY United States

 $\mathbf{DT}$ Journal; Article; (JOURNAL ARTICLE)

LA English

Priority Journals FS

EM9310

Myelin basic protein (MBP)-specific T-cell lines from patients with AB multiple sclerosis (MS) and healthy controls were analyzed for the expression of CD45 isoforms and adhesion molecules. In the multiple sclerosis group, 22 of 24 MBP-specific T-cell lines were CD4+. Two distinct patterns were observed with regard to CD45 isoform expression. Pattern I showed dual expression of CD45 isoforms (CD4+CD45RA+CD45RO+CD29+) and Pattern II included cells with a single CD45 isoform (CD4+CD45RA-CD45RO+CD29+). All 10 cell lines from healthy controls were CD4+ and displayed Pattern II (CD4+CD45RA-CD45RO+CD29+). The dual expression of CD45 isoform in T-cell lines from MS was stable, did not represent a transition stage from CD45RA to CD45RO, and was cell-cycle independent. All cell lines from MS and controls expressed increased levels of LFA-1 (CD11a), LFA-2 (CD2), LFA-3 (CD58), ICAM-1 (CD54), and VLA -4 (CDw49d). These data show the presence of unique MBP-specific T cells (CD4+CD45RA+CD45RO+CD29+) that might play a role in the pathogenesis of MS.

L57 ANSWER 7 OF 11 MEDLINE 1994

AN 93118664 MEDLINE

Cell adhesion molecules in inflammation and thrombosis: status and ΤI prospects.

AU Arnaout MA

CS Department of Medicine, Massachusetts General Hospital, Charlestown 02129.

NC AI-21964 (NIAID)

AI-28465 (NIAID)

Am J Kidney Dis, (1993 Jan) 21 (1) 72-6. SO Journal code: 3H5. ISSN: 0272-6386.

United States CY

 $\mathbf{DT}$ Journal; Article; (JOURNAL ARTICLE) General Review; (REVIEW) (REVIEW, TUTORIAL)

LA English

Priority Journals FS

EM

Cell-cell and cell-matrix interactions play vital roles in AB morphogenesis, inflammation, thrombosis, wound healing, immune surveillance, and growth and metastasis. A number of cell surface HIV-infected cells, and asthma. The antibodies can also be useful in methods of diagnosing and localizing sites of inflammation and tumors expressing ICAM-1. Thus, hybridoma cell line R6-5-D6 producing anti-ICAM-1 antibody was grown and mRNA was extd. for construction of cDNA libraries in Escherichia coli. Colonies pos. for light chains were identified by use of TCCAGATGTTAACTGCTCAC, a probe complementary to a sequence in the mouse .kappa. const. region, and colonies pos. for heavy chains by use of a 980-bp fragment of a mouse IgG2a const. region clone. DNA inserts from these colonies were used to construct expression vectors based on plasmid pEE6-hCMV contg. a polylinker for gene insertion after the major immediate early promoter/enhancer of human cytomegalovirus. The resulting plasmids (pAL5 for light chains and pAL6 for heavy chains) were cotransfected into COS cells which then secreted assembled antibody. Further genetic manipulation produced chimeric light and heavy chain genes (the latter of various isotypes) which were similarly incorporated into expression vectors and expressed in The chimeric antibodies produced were purified by COS cells. affinity chromatog. on protein A-Sepharose. When tested against JY cells (a human B-lymphoblastoid cell line which constiutively expresses ICAM-1 on the cell surface), the chimeric anti-ICAM-1 antibodies showed differences in avidity depending on isotype. The chimeric antibodies inhibited the mixed lymphocyte reaction (an in vitro model of transplantation) and the Schwartzmann reaction (a model of neutrophil-mediated vascular damage assocd. with reperfusion injury and other acute inflammatory disorders).

- L57 ANSWER 10 OF 11 CA COPYRIGHT 1994 ACS
- AN 117:24688 CA
- TI Humanized complementarily-determing region (CDR)-grafted antibodies to intercellular adhesion molecule-1 (ICAM-1), methods of preparation and usage thereof
- IN Adair, John Robert; Athwal, Diljeet Singh; Rothlein, Robert A.
- PA Celltech Ltd., UK; Boehringer Ingelheim Pharmaceuticals, Inc.
- SO PCT Int. Appl., 81 pp. CODEN: PIXXD2
- PI WO 9116927 A1 911114
- DS W: AT, AU, BB, BG, BR, CA, CH, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MW, NL, NO, PL, RO, SD, SE, SU, US
  - RW: AT, BE, BF, BJ, CF, CG, CH, CM, DE, DK, ES, FR, GA, GB, GR, IT, LU, ML, MR, NL, SE, SN, TD, TG
- AI WO 91-US2942 910429
- PRAI GB 90-9549 900427
- DT Patent
- LA English
- The title antibodies are provided, which are useful for treatment of e.g. (non)specific inflammation, rhinoviral infection, human immunodeficiency virus (HIV) infection, the dissemination of HIV-infected cells, and asthma. The antibodies of the invention are also useful in methods of diagnosis and localization of sites of inflammation and infection and ICAM-1-expressing tumors. Recombinant prodn. of the antibodies is described, as is their binding activity.
- L57 ANSWER 11 OF 11 CA COPYRIGHT 1994 ACS
- AN 83:188874 CA
- TI Increased urinary excretion of albumin, light chains, and